STATEMENT OF
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NATIONAL COALITION AGAINST THE MISUSE OF PESTICIDES
ON
PETITION TO THE UNITED STATES CONSUMER PRODUCT SAFETY COMMISSION TO BAN ARSENIC TREATED WOOD IN PLAYGROUND EQUIPMENT AND REVIEW THE SAFETY OF ARSENIC TREATED WOOD FOR GENERAL USE
BEFORE THE
U.S.CONSUMER PRODUCT SAFETY COMMISSION
WASHINGTON, DC

MARCH 17, 2003

Mr. Chairman and member of the commission. I am Jay Feldman, executive director of Beyond Pesticides/National Coalition Against the Misuse of Pesticides (Beyond Pesticides). Beyond Pesticides is a national, grassroots, membership organization that represents the interest of community-based organizations and a range of people seeking to improve protections from pesticides and promote alternative pest management strategies that reduce or eliminate reliance on pesticides. I appreciate the opportunity to speak to you today on behalf of our members.

Beyond Pesticides has worked on the issue of preservative treated wood for over twenty years and has helped promote public awareness of the dangers present in unaware of the toxic chemicals it contains. For this reason strict consumer protection is long overdue. We fully support the petition put forth by the Environmental Working Groups (EWG) and the Healthy Building Network to immediately ban all use of this chemical in playground equipment and to review the safety of arsenic for general use.

Failure to Adequately Regulate CCA

Until now the U.S. Environmental Protection Agency in its roll as pesticide regulators has inadequately protected the health and safety of the American consumer. EPA has a long history of failing to protect the public from the known dangers of CCA treated wood. Therefore, it urgently necessary for the CPSC as an Independent Federal Regulatory Agency to do its best "to save lives and keep families safe by reducing the risk of injuries and deaths associated with [this] consumer product."[1] The best way to decrease these risks is by granting the petition for immediate cancellation of CCA in playground equipment now in front of the commission. EPA has long had the opportunity to protect consumers by regulating the use of CCA treated wood but has failed to do so.

Over twenty years ago EPA knowingly endangered American consumers by promoting inadequate regulation for CCA treated wood. In 1980 Congress exempted

[1] "What we do" from the CPSC website http://www.cpsc.gov/about/faq.html#wha

arsenic treated wood from all hazardous waste laws even if the waste failed the Toxicity Characteristic Leaching Procedure (TCLP), a test created to prevent dangerous materials from being disposed improperly.[2] Even then this wood was known to contain the hazardous and carcinogenic chemicals arsenic and Chromium VI at dangerous levels.

As early as 1978 EPA began reevaluating the registration for CCA because it identified the extraordinary high risk to human health caused by exposure to CCA and other widely used wood preservatives.[3] When the findings were released in 1984, four years behind schedule, unreasonable risk to human health was found, yet registration was maintained. Mitigation measures proposed at the time by EPA were later reduced to become even less protective.

In 1984 EPA issued a press statement describing the decision to maintain CCA's registration and the risk reduction measures it was requiring. It announced a mandatory Consumer Awareness Program (CAP) to ensure all safety precautions were followed. The program required the wood preserving industry to inform consumers about the necessity to use protective gloves, coveralls, and facemasks when sawing treated wood. It stated, "without these restrictions, the risk to public health from using these pesticides would outweigh the benefits." After a series of negotiations with registrants and trade associations EPA deleted the mandatory program in favor of a voluntary one.[4]

This voluntary CAP is largely seen as a failure by both consumer advocates and EPA. At least since 1991, EPA has received reports, through its Office of Pesticide Programs' Incident Data System, of injuries to people exposed to CCA-treated wood, including persistent rashes, eye irritation and neurological symptoms. In 1998, the South Dakota Department of Agriculture (SDDA) conducted a statewide on-site survey of 40 retail lumber yards to determine awareness and compliance with EPA's voluntary CAP. It found that less then 10% of retailers were furnishing consumer information sheets to customers who purchased treated lumber. In response to the notification of the retailer's failure to implement the program, EPA headquarters "indicated that there is a nationwide lack of participation in the voluntary CAP; however they [EPA] are unable to force participation."[5] In 2001 EPA concluded in a public press release that, "the previous consumer awareness program was not adequately informing the public."[6] Despite the known failure of the voluntary program over the last 15 years, and EPA's statement in the 1984[7] and 1986 Rebuttable Presumption Against Registration (RPAR) decision that

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[2] 40 CFR 261.4 (b) under the Resource Conservation and Recovery Act

[3] EPA, 1978. Initiation of Schedule for Review of Wood Preservative Pesticides and Notice of Rebuttable Presumption Against Registration and Continued Registration of Certain Pesticides. 40 CFR 48154

[4] 51 Fed. Reg. 1338 (January 10, 1986)

[5] State FIFRA Issue Research and Evaluation Group, Issue Paper: Elimination of Mandatory Consumer Awarness Program for Creosote, Pentachlorophenol, and Inorganic Arsenical Treated Wood. Presented at SFIREG meeting in Seattle, May 18-19, 1998

[6] Environmental Protection Agency, Headquarters Press Release: Stronger Consumer Information Program, Science Advisory Panel Meeting Announced for CCA-Treated Wood. July 3, 2001. Available at http://yosemite.epa.gov/opa/admpress.nsf/.

[7] "without these restrictions, the risk to public health from using these pesticides would outweigh the benefits." 49 Fed. Reg. 28666

without an effective CAP, the use of pressure treated wood would pose unreasonable adverse effects, and despite its statements that it would adopt a mandatory program if the voluntary program was not successful[8], EPA decided to continue with the voluntary program with some modifications.

In 1988 EPA canceled all non-wood uses of CCA due to concerns of oncogenicity, mutagenicity, teratogenicity, and acute toxicity. EPA's Carcinogen Assessment Group then classified arsenic as a Group A known human carcinogen.[9] In 1993 EPA canceled that last non-wood use of CCA but declined to revise its earlier risk/benefit assessment allowing wood, including for playground equipment and other residential uses, to continue to be treated with CCA.[10]

EPA stated in the 1981 preliminary RPAR that registration of CCA was only maintained due to lack of alternatives.[11] EPA has made no substantial effort to reevaluate these claims since that date over twenty years ago. In reality, tremendous improvements in the availability and economic feasibility of both chemical and non-chemical alternatives to CCA treated wood have been made. In fact, many manufacturers and retailers have already moved away from CCA and into safer alternative materials. Alternatives such as arsenic-free ACQ treated wood, arsenic-free borate treated wood, naturally rot resistant wood such as cedar and redwood, recycled plastic lumber, and wood pulp and plastic composites, are readily available for use in the residential market.

Although EPA published a proposed voluntary cancellation agreement for residential uses of CCA treated wood on February of 2002,[12] one year later the final agreement has yet to be published. This phase out, if released in its initial form, will end production in December of 2003 but will allow this wood to be sold by manufacturers and retailers until all existing stocks are depleted. This agreement will allow children to continue to be exposed to newly treated CCA structures for years to come. It was also reported in the December 20, 2002 edition of the *New York Times* that the proposed CCA phase-out is on a Bush Administration list of 300 federal regulations that may be modified or rescinded in the coming year at the request of industries and consumer groups.[13]

Health Effects of CCA

Health effects of the chemicals in CCA treated wood are well-known. The story of the heavy-duty wood preservatives is a silent tragedy caused by the EPA's failure to

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[8] "Should the mandatory Consumer Awareness Program fail to meet the agencies expectations, the agency is prepared to issue a rule pursuant to the Toxic Substances Control Act directed to alert all purchasers and users of treated wood to appropriate information about the use of such products." 51 Fed Reg. 1334 (Jan. 10, 1986)

[9] 53 Fed. Reg. 24787, 89 (June 30, 1988)

[10] 58 Fed. Reg. 64581 (December 9, 1993)

[11] 46 Fed. Reg. 13032

[12] 67 Fed. Reg. 8244-46 (February 22, 2002)

[13] Seelye, Katharine, White House Identifies Regulations That May Change, *New York Times,* December 20, 2002

act on the side of caution, failure to embrace the precautionary principle for the protection of children, and failure to enforce the unreasonable adverse effects standard of the *Federal Insecticide Fungicide and Rodenticide Act* (FIFRA). Let us not forget that childhood cancer is one of the leading and least understood causes of childhood death. A child born today faces a risk of 1 in 600 of falling ill to cancer by the age of ten. Inorganic arsenic and hexavalent chromium (Cr (VI)) are classified by the U.S. Environmental Protection Agency as a "Known Human Carcinogen"(Group A)[14]. As a carcinogen, any exposure increases the lifetime risk of cancer.

There is evidence that chronic toxicological effects of arsenic can occur at doses as low as 0.15 mg daily. Many health impacts clinically linked to arsenic exposure such as high blood pressure, irregular heartbeat, premature hardening of the arteries, and anemia are common throughout the population and may not be easily linked to long-term low-level exposure to arsenic.[15] Arsenic is found in many public water sources throughout the country, these exposures are a major public health threat, therefore any additional exposures must be vigorously limited, especially those that effect children.

Studies of Cr (VI), from industrial emissions, have found it to be highly toxic due to strong oxidation characteristics and ready membrane permeability.[16] Cr (VI) has been known to cause damage to kidneys and liver. Birth defects have been observed in animals exposed to chromium (VI). Skin contact with certain chromium (VI) compounds can cause skin ulcers. Some people are extremely sensitive to chromium (VI) or chromium (III) and allergic reactions consisting of severe redness and swelling of the skin have been noted.[17]

New Exposure Studies Call for CSPC Action

Low dose neurological effects are well-documented with arsenic exposure. There is evidence that low-dose exposure to arsenic can have human health impacts. Although past studies have concluded that neurological function was not impaired below 1000 ppb, a recent EPA study finds that vibrotacitle and pin-prick sensitivity were affected at levels as low as 300 ppb in drinking water.[18] Low dose arsenic exposure has also been statistically linked to height in growing children. A Thailand Health Research Institute study shows an inverse relationship between the levels of arsenic found in children's hair and their height. This relationship was significant for both high and low arsenic accumulations. This study represents defining data on low-level arsenic exposure's effect

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[12]Pesticide.Net, 1999, Pesticidal Chemicals Classified as Known, Probable or Possible Human Carcinogens, http://www.pestlaw.com/x/guide/1999/EPA-19990100A.html

[15] Morton, E., Dunnette, D., 1994, "Health Effects of Environmental Arsenic", *Arsenic in the Environment, Part II: Human Health and Ecosystem Effects* Jerome Nriagu, editor John Wiley & Sons, Inc

[16] Hazardous Substance Data Bank (HSDB), National Library of Medicine Specialized Information Service http://toxnet.nlm.nih.gov/cgi-bin/sis/search

[17] Agency for Toxic Subsistances and Disease Registry, U.S. Center for Disease Control, http://www.atsdr.cdc.gov/tfacts7.html

[18] Mumford, Judy, PhD, Yajuan Xia, Mike Schmitt, Richard Kwok, Zhiyi Liu, Rebecca Calderon, David Otto, *Health Effects from Chronic Exposure to Arsenic via Drinking Water in Inner Mongolia*, EPA Human Studies Facility, Research Triangle Park, NC

on the growth of children.[19] The Minnesota Arsenic Study (MARS), conducted by the Minnesota Department of Health (MDH), finds that children accumulate arsenic at a higher rate then adults.[20] This finding places more urgency on the Commission to weigh the evidence, with children and their unique physiological chemistry in-mind. The limits of low-dose exposure to arsenic, especially when dealing with children's exposure must be padded with an extensive safety factor even with limited evidence. Unlike most chemicals, we cannot rely on animal data to interpret possible human health effects due to the well-known human sensitivity to inorganic arsenic.

Soil and Surface Residues

The amount of exposure children have to the toxic chemicals contained within this wood is of vital importance to these hearings. Hand-to-mouth behavior among children is well documented and can significantly contribute to this chemical exposure. Exposure can come from direct contact to the CCA wood or from contacts with contaminated soil. This behavior is especially significant because of arsenic's low rate of dermal absorption and high rates of absorption through ingestion. According to the October, 2001 EPA Scientific Advisory Panel (SAP) recommendation, children have an average of 9.5 hand-to-mouth activities per hour for an average of 1-3 hours of play activity.[21] This number has the potential to grossly underestimate the true exposure to active children. This is especially pertinent to high-risk groups who are predisposed to high rates of hand-to-mouth behavior, such as children with Down syndrome (DS).

Leaching into soils surrounding and under CCA-treated structures is well documented. A community group in Ithaca, NY found soil samples under a CCA treated playset with levels of arsenic up to 101 parts per million (ppm), over ten times the New York state clean-up standard of 7.5 ppm.[22] Arsenic levels averaging 76 ppm under CCA treated decks compared to an average level of 3.7 ppm of arsenic in control soils was reported by the Connecticut Agricultural Experiment Station.[23] The FIFRA October 2001 SAP used a 25% availability factor for arsenic of consumed soils,[24] this factor does not take into account the variability of soil types with differing pH and organic content in the diverse area of CCA treated wood usage in the U.S.. In soils with differing pH levels this rate could be even greater.

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[19] Siripitayakunlit, Unchalee, Amara Thonghong, Mandhana Pradipasen, 2000, *Growth of Children with Different Arsenic Accumulation, Thailand*, University of Denver Poster, financed by the Thailand Health Research Institute, National Health Foundation

[20] Minnesota Department of Health (MDH), 2001, *The Minnesota Arsenic Study (MARS), http://www.health.state.mn.us/divs/eh/hazardous/arsenicstudy.pdf*

[21] FIFRA Scientific Advisory Panel, 2001, *Final Expo Document October 23-25,* http://www.epa.gov/scipoly/sap/2001/october/final_expo_doc_927.pdf, p. 15

[22] Steingraber, S; Arsenic and Old Spaces, Pesticides and You, Reprinted Op-ed

[23] Stillwell, D., and Gorny, K, 1997, Contamination of soils with copper, chromium, and arsenic under decks built from pressure treated wood, Bulletin of Environmental Contamination Toxicology, 58(22-29)

[24] FIFRA Scientific Advisory Panel, 2001, *Final Expo Document October 23-25,* http://www.epa.gov/scipoly/sap/2001/october/final_expo_doc_927.pdf, p. 15

Additional exposure to the chemical constituents of CCA can come from the practice of CCA wood in gardens in residential settings. David Stilwell found that when growing lettuce in gardens with CCA treated wood blocks; the lettuce's arsenic uptake was more then 1.7 ppm arsenic by dry weight.[25] These levels alone may not cause acute poisoning but when considered in as an additive to other exposures they are far from negligible.

Wipe test studies to determine the amount of dislodgeable arsenic in CCA treated wood conducted by the Connecticut Agricultural Experiment Station found that dislodgeable arsenic varies greatly depending on age and use. Wipe tests done on the horizontal surfaces of three municipal play structures found an average of 8.8 µg/100 cm^2 of arsenic dislodged from the wood.[26] Tests done on the vertical support beams of the structures found higher levels of arsenic compared to the horizontal surfaces and the new samples. These levels were as high as 632 µg/ 100 cm^2 and averaged 105 µg/100 cm^2.

Conclusion

In conclusion, with the evidence presented here today along with the findings of the Commission's own report on the risk of cancer from children's exposure to CCA-treated playground equipment, there can be only one conclusion: the CPSC must immediately grant the petition in question and ban the use of chromated copper arsenate in the production and sale of children's playground equipment. Please carefully consider the evidence presented here on the inability and unwillingness of EPA to properly ensure the safety of America's children and consumers in general, over a twenty five year period, EPA has known the dangers of this product. Each additional day guarantees more children will suffer harm. The Commission cannot defer to the authority of the U.S. Environmental Protection Agency in this matter, and must move to carry out its statutory mandate to protect the safety of children now.

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[25] Stilwell, David, 1999, Arsenic in Pressure Treated Wood, Department of Analytical Chemistry, The Connecticut Agricultural Experiment Station,

[26] Stilwell, David E., 1998, Environmental Issues On The Use Of CCA Treated Wood, Department of Analytical Chemistry, The Connecticut Agricultural Experiment Station, http://www.caes.state.ct.us/FactSheetFiles/AnalyticalChemistry/fsAC001f.htm

ACCA Ban Petition, Petition HP 01-3@

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February 28, 2003

Re: ACCA Ban Petition, Petition HP 01-3@

I am here today to serve as an advocate for a subset of children who exceed your 1 in 10,000 risk assessment for cancer. This particularly vulnerable group of children requires protection and I will not stop until they get the recognition they deserve. The group I am referring to is special needs children.

Roughly one out of every 500 children born has Trisomy 21, better known as Down syndrome. My daughter, Emily, was born with Down syndrome. Anyone who has spent time with Down syndrome children knows that they experience their world through their mouths well beyond the oral stage seen in typical children. Emily at age 8 still mouths many objects and uses her hands to play with her protruding tongue.

We had frequently observed Emily putting her tongue directly on our wooden deck or chewing on the railing. Like many other parents, I was unaware that CCA wood contained toxic metals. We became painfully aware of this when we sanded our deck and Emily suffered neurological problems from an episode of acute arsenic poisoning.

I submitted a written letter to the school nurse forbidding Emily to play on the CCA playground. While my request was accommodated, they continued to take the rest of the special needs children to the playground despite my efforts to inform them of the increased risk our very "oral" children have.

What could a mother possibly know? Surely if there was any danger to our special children, someone of "authority" would notify us.

I would like to share with you exactly what someone who is "just a mother" has learned.

Your study looking at the long-term risk of cancer from CCA exposure during childhood is a step in the right direction; however, with childhood cancers on the rise one needs to consider the impact of environmental toxins such as CCA on childhood cancer. A recent

study is quoted as saying, "**Acute lymphoblastic leukemia (ALL) is the most common pediatric cancer. The origin of this disease can be explained by a combination of genetic susceptibility factors and environmental exposures. For the purpose of our study it can be considered as a complex disease, caused by the "carcinogenic" effect of the environment modified by a series of genes**" (1)

Down syndrome children have a 15-20 fold increase occurrence of leukemia when compared to non-trisomic children. While Emily's genetics may have set the stage for cancer, I believe her chronic exposure to CCA lumber was the trigger to her leukemia. What follows is my reasoning.

Down syndrome(DS) chemistry and CCA exposure; A leukemia connection

Fact number one: Due to their genetics DS individuals overproduce hydrogen peroxide; arsenic exposure increases hydrogen peroxide. This causes DNA damage and increased risk for leukemia.

...excessive hydrogen peroxide production in Down syndrome

The gene for Cu-Zn superoxide dismutase (SOD) is coded on chromosome 21 which is overexpressed in DS. Such overexpression results in increased oxidative stress (an increase in ROS) and an overproduction of hydrogen peroxide (H2O2) in DS individuals.(2-4)

...excessive hydrogen peroxide production with arsenic exposure

Exposure to arsenic increases SOD activity (5) and results in excessive hydrogen peroxide production and oxidative stress (6)

Clearly, arsenic exposure exacerbates the genetic overexpression of SOD and compounds the already burdened oxidative stress seen in Down syndrome. A study which looked at hydrogen peroxide induced DNA damaged states,

"The mechanism of carcinogenesis in Down syndrome could be explained by our findings: SODs enchance metal-mediated DNA damage induced by H2O2. ...We conclude that SODs may increase carcinogenic risks, e. g. of tumors in Down syndrome."(7)

Oxidative damage is especially true in the presence of copper. Excessive copper levels have been reported in DS (8,9). Erythrocytes, thrombocytes and neutrophils of DS individuals were found to have significantly higher levels of copper.(9)

It has not yet been determined if copper leaches from CCA wood in the same way that arsenic does; however, animal studies show high copper levels can interfere with arsenic excretion. Delayed excretion would increase risk for arsenic toxicity.

In a study which looked at DNA damage from lead or mercury, it has been conclusively demonstrated, "that there is a causal relationship between the induction of H2O2 and the mutagenic potential of these metals." (10)

Arsenic acts the same as lead or mercury in its excessive production of hydrogen peroxide and related DNA damage.

Fact number two: DS individuals have poor DNA repair mechanisms; arsenic exposure inhibits DNA repair. If DNA damage is unable to be repaired this may initiate leukemia.

...poor DNA repair in DS

It is well documented that DS cells have a profound DNA repair deficiency. "The same features apply for cells, which contain an overexpressed Cu/Zn-superoxide (SOD-1) gene." (11) Two types of DNA repair deficiencies have been noted; a "DNA repair deficiency in strand break repair and also a second DNA repair deficiency in incision activity." (12)

It has been proposed that, "This altered repair system is probably responsible for the increased frequency of chromosome aberrations that can be induced in these cells by x-rays and the increased tendency for leukemia observed in Down syndrome as well." (13)

...arsenic exposure inhibits DNA repair

Once again arsenic exposure compounds the genetic problems seen in DS. Arsenic exposure has been documented to cause DNA repair deficiency.(14-16) A newly published study shows that arsenic "interferes with various DNA repair systems in concentrations in the low micromolar range." (16) More specifically poly(ADP-ribosyl)ation has been shown to be "inhibited at concentrations as low as 10nM...Since poly(ADP-ribosyl)ation is an immediate cellular response to DNA damage, playing a major role in DNA base excision repair and the maintenance of genomic stability, its inhibition by arsenite may add to the risk of cancer formation under low-exposure conditions." (16) This is bad news for DS where it has been demonstrated that "DS lymphocytes are more sensitive to the inhibition of poly(ADP)ribose synthetase than normal lymphocytes."(17)

How bad is the compounding effect of DS genetics and arsenic exposure?

One sudy states, "...chromosome rearrangements may exist in proliferating cells in DS individuals after exposure to clastogens and that this abnormality predisposes them to develop leukemia."(18) Of course arsenic has been shown to be a potent clastogen so it can be viewed as a trigger to leukemia in a genetically vulnerable population such as DS.

Fact number three: DS individuals have low plasma glutathione; arsenic exposure can deplete glutathione. Epidemiologic evidence suggests low glutathione is a risk factor for leukemia.

...plasma glutathione (GSH) in Down syndrome

The Nutrition-Environment Interactions Research Core Group at the University of California at Berkeley "has uncovered epidemiologic evidence that lower intakes of certain amino acids are associated with increased risk of childhood leukemia. This research suggests that deficiency of gluthathione, a major plasma antioxidant, and its precursor, cysteine, may increase the risk of this disease." (19)

A recent study found the plasma glutathione (GSH) levels to be significantly reduced in children with DS.(20) Animal models of DS have shown decreased GSH in cultured hippocalmpal neurons which contributes to cell loss and neurodegenration in DS.(21) Some Down syndrome cells have been shown to have an adaptive mechanism of upregulating glutathione peroxidase (GPx) in response to the SOD overexpression.(22) GPx activity remains unaltered in DS brains (22) and lymphocytes (23) thereby leaving these cells more prone to oxidative injury from excessive H2O2. Thus you can expect to see increased neurotoxicity in addition to DNA damage in DS individuals exposed to CCA.

...glutathione (GSH) in arsenic exposure

A recent study found some human cells could develop tolerance to chronic arsenic exposure. The tolerant cells "had increased basal GSH levels (4.9-fold) and increased GST activity (2.4-fold) and both GSH depletion and inhibition of GST activity abolished arsenic tolerance.....Our results indicate that this tolerance in human cells involves increases in GSH levels and GST activity that allow for more efficient arsenic efflux..."(24)

The ability of arsenic to perturb cellular glutathione regulation is highly tissue specific. For example in one study GST activity was shown to increase in keratinocytes but not in fibroblasts or breast tumor cells. (25) Studies need to be tissue specific and in the case of CCA exposure include simultaneous exposure to copper and chromium.

Again we find that a DS child would be much more affected by the damages of arsenic than a non-genetically predisposed child.

Fact number four: DS individuals have altered folate and methylation cycles; arsenic exposure perturbs these same cycles. Alterations in folate have been implicated in leukemia.

...altered folate and methylation in Down syndrome

Another gene localized to chr 21 is cystathionine Beta-synthase (CBS). CBS catalyzes the conversion of homocysteine to cystathionine. A consequence of the CBS over expression is a reduction of homocysteine available for remethylation to methionine. Thus in DS you find reduced plasma levels of homocysteine, methionine, S-adenosylmethionine (SAM) and S-adenosylhomocysteine (SAH).(20) Folate trapping and a functional folate deficiency are established. This is reflected in the elevated MCV and increased sensitivity to the antifolant drug methotrexate found in the DS population. The folate trapping caused by CBS overexpression results in thymidylate deficiency and the consequential misincorporation of uracil. This in turn requires that the DNA repair mechanism be working optimally; a condition that arsenic exposure even in low doses prevents.

...altered folate and methylation in arsenic exposure

The resynthesis of GSH under conditions of GSH depletion has been shown to cause a decrease in methionine and impairment in DNA methylation.(26,27) Plasma methionine levels in DS children were found to be 53% of that for control children.(20) Therefore the GSH depleting properties of arsenic amplifies the problem of CBS overexpression associated with DS. Additionally, arsenic has been shown to down regulate DNA methyltransferase1. (5)

GSH depleting agents cause a diversion of folate away from the biosynthesis of purines and the pyrimidine thymidylate.(26)

"Thus alterations in gene expression could result from a high dose and/or prolonged exposure to GSH-depleting agents, e.g. medications, chemotherapeutic agents and environmental toxins."(27)

This leaves DS individuals who have a functional folate deficiency and low plasma GSH levels at increased risk of cancer upon exposure to agents such as those found in CCA wood. This risk exceeds that of non-trisomic individuals.

A multitude of current research has implicated disruptions in the folate cycle with the onset of leukemia. (28-32)

Arsenic causes disruptions in these same pathways(33-37) thus leukemia should now be considered among the cancers induced by environmental exposure to CCA wood in genetically susceptible children.

Dietary Folate Deficeincy Enhances Induction of Micronuclei by Arsenic in Mice (33)

Mouse peripheral blood MN assay of polychromatic erythrocytes (PCE) was used to measure damage to the bone marrow. Mice were made folate deficient. The folate deficient mice had small increased levels of micronuclei before they were exposed to arsenic.

Upon exposure to arsenic, the folate-deficient animals exhibited higher MN-PCE frequencies than the folate-sufficient animals. At the highest dose of arsenic used, the MN-PCE levels in folate deficient animals was 2.4 fold higher than that found in the folate sufficient animals.

"the difference in MN-PCE levels between folate-deficient and folate-sufficient mice treated with arsenic was greater than the sum of the effects of folate deficiency and arsenic considered separately."(33).

The type of chromosomal damage was almost completely due to chromosome breakage.

"Because humans are more sensitive than mice to the genotoxic effects of both arsenic and folate deficiency, the potential enhancing effect of folate deficiency on arsenic genotoxicity in exposed human populations may be greater than that observed in mice. Although further work is needed to address the effects of moderate to marginal folate deficiency and chronic arsenic exposure at lower doses, our results give support to the ideas that dietary folate deficiency may be a predisposing factor in arsenic genotoxicity and carcinogenesis in individuals consuming nutritionally inadequate diets."(33)

DS or other genetic polymorphisms can be the equivalent of "consuming nutritionally inadequate diets". An example of this is seen in the reported increase neurotoxicity of arsenic in persons with methylenetrahydrofolate reductase deficiency.(36)

Fact number five: An abberant response to a viral infection has been proposed to contribute to leukemia risk. DS individuals have altered immune systems; arsenic exposure compounds this problem.

...immune function in Down syndrome

There are many published studies citing the immune deficiencies of Down syndrome individuals. Immune supporting nutrients are also found to be altered in DS.

"Serum zinc and selenium levels were significantly lowered in DS subjects, whereas copper levels were elevated. Consequently a marked increase (40%) of the copper/zinc ratio in DS persons was observed"(38)

...immune function in arsenic exposure

Toxicity of arsenic is partially mitigated by zinc and selenium so here again this environmental toxin is particularly toxic in DS. Zinc deficiency causes a shift from cell-mediated immunity (Th1) to a humoral immunity (Th2).(39,40) Such a shift leaves individuals more sensitive to viral infection. Heavy metal exposure and low GSH levels have also been shown to cause a shift to Th2 thereby increasing susceptibility to viral infections. GSH depletion allows for better replication of viruses while at the same time causing them to mutate to a more virulent form. (41-43). This is a key point; it is against the backdrop of chronic metal exposure that one can have complications with relatively benign viral exposures.

"Research shows that arsenic is a general gene inducer. Genes induced are involved in proliferation, recombination, amplification and the activation of viruses."(44)

If an aberrant response to a viral infection is a contributor to leukemia risk, then it is obvious that arsenic can cause such an aberrant response.

Fact number six: the preleukemic condition prior to development of Acute Lymphocytic Leukemia (ALL) is consistent with that produced by arsenic exposure.

...bone marrow dysfunction preceding ALL

A transient aplastic anemia recovering spontaneously within a few days or weeks is reported to be a preleukemic condition to ALL. The "remission lasts from a few weeks to several months and is followed by overt ALL."(45)

For my daughter, macrocytic anemia (elevated MCV above that which is seen in DS) was picked up on a standard blood test 10 months prior to her diagnosis with ALL. Lab tests were run to determine the underlying cause. No cause could be found. Her liver enzymes were slightly elevated. Hepatitis screening came back negative. I was told there was no need for concern and no further testing was done to investigate the cause. Looking back now, I know that an elevated MCV with a normal RDW is a preleukemic condition (46) and I should have pushed for an investigation to determine the cause.

...hematological effects of arsenic exposure

"The heavy metals most commonly associated with hematologic toxicity are arsenic and its derivative arsine, copper..." (47) Here we see 2 of the 3 CCA metals listed as those which are most commonly associated with hematological manifestations.

Both aplastic anemia (47) and macrocytosis (48) (like the unexplained macrocytosis seen in my daughter prior to her leukemia) are found in arsenic exposure. As stated above these are both preleukemic conditions. A case history of acute myelogenous leukemia after arsenic induced aplastic anemia is reported in the medical literature. (49)

An animal model of arsenic induce leukemia states, "The arsenic-induced leukemias observed showed the whole spectrum of forms known in human pathology." (50)

A recent leukemia study found an elevated risk for leukemia with exposure to arsenic in drinking water. (51) It is once again important to bear in mind that copper and chromium were not included in that study.

Fact number seven: there are miscellaneous other bits of evidence to support a CCA leukemia connection.

...epidemiological studies

"From the epidemiological studies, there is suggestive evidence that hexavalent Cr causes increased risk of bone, prostate, lymphomas, Hodgkins, leukemia, stomach, genital, renal, and bladder cancer, reflecting the ability of hexavalent chromate to penetrate all tissues in the body." (52) While the main focus has been on arsenic exposure from CCA wood, little has been done to determine the bioavailability of the toxic chromium.

In another study some of the factors found to have **elevated and/or significant ORs include**: **Down syndrome**, MMR vaccination [OR = 3.7 for pre-B ALL], measles, **metal exposure** [OR = 2.0 for pre-B ALL] , exposure to insecticides [OR = 2.0 for pre-B ALL], **exposure to dust (most commonly recorded as wood dust)** [OR = 8.0 for pre-B ALL] ect...(53)

What would be the OR and probability for my daughter with Down syndrome exposed to the heavy metals arsenic and chromium from wood dust* known to be derived from CCA treated wood?

*The study that found such a large OR for exposure to dust (most frequently recorded as wood dust) does not go far enough in stating if the wood dust was from chemically treated wood such as CCA).(53)

Aneuploidy, a condition which is found in cancer cells, has been demonstrated in mouse bone marrow cells after exposure to arsenic. (54)

The cancers most commonly quoted as being associated with arsenic exposure are lung, bladder, liver, kidney and skin. That list is by no means exhaustive. This is clearly demonstrated by a study looking at disease in areas with high levels of arsenic in drinking water. Increase mortality from the following cancers were included: larynx, colon, stomach cancers, rectal cancer, and lymphoma. (55) New associations will be made as science progresses.

Lastly, the paper I think is the most important to understand and should be read by anyone considering an arsenic leukemia connection is number 29 on my reference sheet. That paper combined with a good understanding of Down syndrome genetics and the effects of arsenic exposure clearly point a finger at CCA exposure and leukemia in Down syndrome children.

Closing

So what does a person who is "just a mother" know about CCA exposure? I know that we have not done enough to protect our environment and our most valued gift; our children!

Thank you for taking the time to consider my ideas. I am most appreciative of your willingness to take a stand on this serious health issue and to be on the side of the consumer.

Laurette Janak

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March 17, 2003

Presentation of Louis Sullivan, M.D. [1]

———Thank you, Mr. Chairman, for the opportunity to share my views on this important topic this afternoon.

In August 2002 [2], I addressed the debate concerning the safety of CCA treated wood. At that time, I concluded that children are safe, and that play structures constructed of this material have not been shown to pose a risk to health or safety.

This position was supported by the EPA, which stated that it, "does not recommend that consumers replace or remove existing structures made with CCA treated wood or the soil surrounding those structures." The State of Florida formed an expert panel of physicians to evaluate risks associated with CCA treated wood. This group "agrees with and supports" the EPA's position. As I explained at the time, the Florida physicians group also concluded that CCA treated wood has never been linked to increased risk of cancer, which, it concluded, "would be expected after 30+ years of use if toxic levels of arsenic were leaching from the wood."

The staff of this Commission recently issued a report which contains mathematical projections which purport to demonstrate that children who play on CCA treated play structures may face an increased risk of cancer. This report does not alter the conclusion I reached last August. That is, public health judgments must be based on research and evidence and the evidence does not show that CCA treated wood play structures are unsafe.

The Staff Report estimates potential arsenic exposure from treated wood play structures, highlighting the absence of any actual bio-monitoring data. Even if one accepts these theoretical exposures, they are well within the background levels to which most people, including children, will be exposed from food and drinking water. The CPSC Staff Report recognizes this. Neither these potential exposures, nor the theoretical risk derived from the Staff Report's calculations, warrant action by this Commission.

I understand that this product is being withdrawn from use in new play structures, and that EPA already is engaged on this issue. In light of these facts, it is important to ask whether any further activity by this agency is needed.

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[1] President emeritus of Morehouse School of Medicine, Atlanta, Georgia; Served as Secretary of the United States Department of Health and Human Services from March 1989 through January 1993; Serves as a member of the Board of Directors of BioSante Pharmaceuticals, Inc., Bristol-Meyers Squibb Company, CIGNA Corporation, Endovascular Instruments, Inc., Equifax Inc., Georgia-Pacific Corporation, a treater and seller of pressure-treated wood, Minnesota Mining and Manufacturing Company, and United Therapeutics, Inc.; Serves on the Boards of Medical Education for South African Blacks, Project Hope, Africare, and the Little League Foundation.

[2] At that time, I was a medical adviser to the Treated Wood Council.

I believe this Commission could best serve the public by focusing its Staff and limited resources on addressing children's health and safety issues that pose documented threats. The Commission has done good work in minimizing the hazards from many products, such as flammable children's sleepwear, and choking hazards associated with toys. It should continue to focus on projects in which it can make a real difference, rather than in areas that have not been shown to pose a risk in real life.

Because the Surgeon General of the U.S. Public Health Service has identified being overweight as one of the major health problems of American children today, the Commission should be especially careful not to unnecessarily alarm parents and children, so that they avoid physical activity in playgrounds. Such an outcome from addressing a theoretical problem could exacerbate a real, and increasing, one.

In closing, I would urge the Commission to continue its good work by focusing on those issues that pose a real threat to the health and welfare of America's children. The present concerns about treated wood play structures do warrant more study, but there does not appear now to be a public health reason for the Commission to grant the petition that is before it.

Comments on CPSC's analysis of cancer risk to children from contact with CCA-treated wood products

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My name is Kenneth Brown. I hold a Ph.D. in mathematical statistics from Johns Hopkins University. I have published numerous articles on statistics and applications to risk assessment. With respect to arsenic in drinking water, I have served on committees (NRC/NAS subcommittee on arsenic in drinking water, Arsenic Task Force of the Soceity for Environmental Geochemistry and Health), workshops on research needs (NCI/NIEHS/EPA, American Water Works Association), drafted the position paper of the American Council on Science and Health, presented invited and contributed papers at numerous conferences, and co-authored 12 articles - 5 in conference proceedings and 7 in refereed journals, including 2 which were invited. Research has been supported by U.S. EPA, industry, and trade associations (e.g., American Water Works Association).

The CPSC claims that it has dealt with sources of uncertainty and variability, but that is not quite accurate. They have considered some sources of variability within the context of their analysis, which is laudatory, but "uncertainty refers to lack of knowledge in the underlying science" (NRC2, 01, p.109). The CPSC staff needs to consider the credibility of some of its assumptions, which are sources of uncertainty to be identified and addressed openly. Questionable assumptions that are made with good faith, but little or no discussion, tend to lead the trusting but unwary reader to an unfounded level of comfort with the validity of the analysis. Two such assumptions will be discussed. The first is CPSC's equating limited and intermittent exposure to arsenic in the first few years of life to a chronic lifetime low-level exposure with equivalent total arsenic intake. The second concerns the NRC risk assessment where all persons within a given village were treated as if exposed to the same concentration of arsenic in drinking water, specifically the median concentration of wells tested within the village. Both assumptions are the result of genuine limitations of the science, or the available data, but they still undermine the credibility of the NRC risk assessment and CPSC's extrapolation of its results to estimation of children's risk from contact with CCA-treated wood.

The first assumption implies that estimated lifetime cancer risk is based only on total cumulative lifetime arsenic intake, regardless of how it is distributed over the lifetime. The Taiwan data, however, don't support that assumption. Although flawed with regard to exact exposure levels, the data indicate that duration of exposure (number of years) is more important than daily intake in determining cancer risk. This suggests that long-term exposure, or an unexplained age effect, is having a substantial impact on estimates of lifetime risk of cancer. Neither would apply, however, for arsenic exposure during childhood alone. It follows that the effect of the assumption made in the CPSC procedure

would be to overestimate the effect of early-life exposure to arsenic on lifetime cancer risk. It is not clear,however, that even *lifetime daily intakes* at the low arsenic levels experienced by children coming into contact with CCA-treated wood would pose an increased cancer risk.

The principal argument for low-dose linear cancer risk, and hence risk at extremely low arsenic concentrations, as assumed in the NRC reports and by the CPSC staff, is more a matter of policy than science. Arsenic does not appear to act directly on DNA, the main argument for low-dose linearity. As noted in NRC1 (p.7), "Of the several modes of action that are considered most plausible, a sublinear dose-response curve in the low-dose range is predicted, although linearity cannot be ruled out". Arsenic is ubiquitous and is possibly even beneficial in small quantities. Such evidence is indirect, based on animal experiments that found arsenic may be nutritionally essential (the two NRC reports emphasized the lack of direct evidence for humans but the EPA risk assessment forum of 1986 (EPA, 1986) considered it more seriously). The point is that even if the risk at very low concentrations in drinking water were reliable (to be discussed next), extrapolation of risk estimates based on chronic exposure to children who are intermittently exposed to CCA-treated wood in childhood is speculative. As a practical example, the assumptions being made in the CPSC analysis about the risk from arsenic would not apply, for example, to tobacco smoke. Tobacco smoke contains hundreds of compounds including at least 40 known carcinogens, and one might speculate that they would probably cover most modes-of-action for chemical carcinogenesis. It is known, however, that the risk of lung cancer diminishes with time, almost to that of a never-smoker, if exposure (smoking) is terminated.

The second assumption to be discussed concerns the uncertainty in the risk assessments of the NRC and U.S. EPA for cancer from arsenic in drinking water. There is uncertainty in all risk assessment, but in this case it was assumed that all the study subjects within a given village were exposed to the same arsenic concentration in drinking water, i.e., they were treated as if they all drank from a single source with the arsenic concentration at the median value of the wells tested within the village. Wells within the same village, however, often differed dramatically in arsenic concentrations. Figure 1, showing the arsenic concentrations by village, for villages with more than one well, was constructed from Table A10-1 of the first NRC report (NRC1, 1999). The same data were analyzed more fully in the article by Morales et al. (2000) that was cited heavily in the second NRC report and in the EPA report.

The first village listed in Figure 1, O-G, had a relatively large number of cancer occurrences. All the recorded cancers for the village were treated as having occurred at exposure concentrations of 30 μg/L. There were five wells, however, tested at 10, 10, 30, 259, and 770 μg/L. What is missing from the data is the distribution of the population in the village across wells, i.e., how many used each well, and, the distribution of the cancer cases across wells, i.e., the number of cancer cases at each well concentration. Not all villages are so extreme, but it is readily apparent from the table that the example just described is not an isolated case. The potential for serious exposure misclassification is obviously high. The effect of such data on risk estimation is apparent in a diagram in

which different dose-response models were fit to the data. First, however, it maybe useful to see an example of a model fit to good dose-response data.

The data in Figure 2 are from mortality of rats exposed to hydrogen sulfide, and are used here strictly for illustration, with a logistic model fit to the data. A statistical measure of the goodness of fit, or something such as the AIC (Akaike Information Criteria) used by the NRC to compare different alternatives, is not adequate by itself; it is necessary to graphically examine the fit of the data. In this case, it is apparent graphically that the model describes the data well – the data are close to the curve and predicted values calculated from the curve should be reasonable. Another model might fit the data about equally well, but to do so it is clear that it would have to be very close to the current curve. Thus one can have some level of comfort in using the fitted curve to estimate risk at arbitrary exposure values that may not have been actually observed.

By contrast, several different models were statistically fit to the Taiwan data, using no comparison population (a choice favored by EPA and the EPA Science Advisory Board), and using either the southwest region of Taiwan or all of Taiwan as a comparison population for the study area (NRC2 favored the southwest region). The results are displayed in Figure 3, which appeared in Morales et al. (2000) and NRC2. It is clear that the data are so variable that none of the models provide a good fit to the data. One point near the center of the exposure range is exceedingly high, suggesting that it might be an outlier. More than one model has about the same AIC value, indicating that they cannot be distinguished on a statistical measure of fit (the AIC provides a relative comparison of fits – no statistical measure of fit was found). As one can see graphically, the estimated risks very close to the origin vary widely for different models, so there is considerable model sensitivity.

Nevertheless, the NRC settled on one of the models using the southwestern Taiwanese region as the comparison group, and concluded that the model "provides a satisfactory fit to the epidemiological data and represents a reasonable model choice for use in arsenic risk assessment" (NRC2, p.175). It is hard to see how that statement would be justified even if the data were reliable. Given what is undoubtedly a high error rate in exposure classification in the data, there would be little basis for much credence in any model fit to the data.

What is the NRC's conclusion about the Southwest Taiwan database? That depends on whether you read NRC1 or NRC2, and whether you read the executive summary or the body of the report. The NRC2 executive summary states "There is a sound database on the carcinogenic effects of arsenic in humans that is adequate for the purposes of a risk assessment." The NRC1 executive summary, however, makes the recommendation that "Additional epidemiological evaluations are needed to characterize the dose-response relationship for arsenic-associated cancer and noncancer end points, especially at low doses. Such studies are of critical importance for improving the scientific validity of risk assessment." In the body of NRC1, it is noted that "in some cases, arsenic concentrations varied considerably in different wells within the same village (see Addendum). Hence, there is considerable uncertainty in the data" (p. 274). Morales et al. (2000) commented

that exposure is measured at the village level, and that there appears to be variability in the exposure assessment, causing high variability in the risk estimates.

Of the two sources of uncertainty described above, the first addressed an assumption that CPSC needed to make to extrapolate cancer risk estimates based on chronic exposure to intermittent childhood exposure from contact with CCA-treated wood products, given that the estimates for chronic exposure to low arsenic concentrations in drinking water are valid and reliable. The second source of uncertainty addressed the limitations of the data for making valid and reliable estimates at low arsenic concentrations in drinking water. The value of CPSC's objective is not in question, but it is unrealistic in view of limitations regarding epidemiological data and the mode-of-action of arsenic carcinogenicity.

As an aside, I drafted a position paper on the risk of cancer from arsenic in drinking water in the U.S. for the American Council on Science and Health. They had it heavily reviewed and then wrote their own conclusion. It was submitted, by invitation, to *Regulatory Toxicology and Pharamacology* where it was peer reviewed again before publication. The conclusion, with which I agree, is that at several hundred µg/L there is clear evidence of cancer and non-cancer effects, but at or below 50 µg/L, limitations regarding the epidemiological data and the mode-of-action of arsenic toxicity are inadequate to support the conclusion that there are adverse health effects in the United States. The implications for the CPSC analysis is that they are trying to ferret out cancer risks at extremely small arsenic intakes for which it is not at all clear that there even is a cancer risk.

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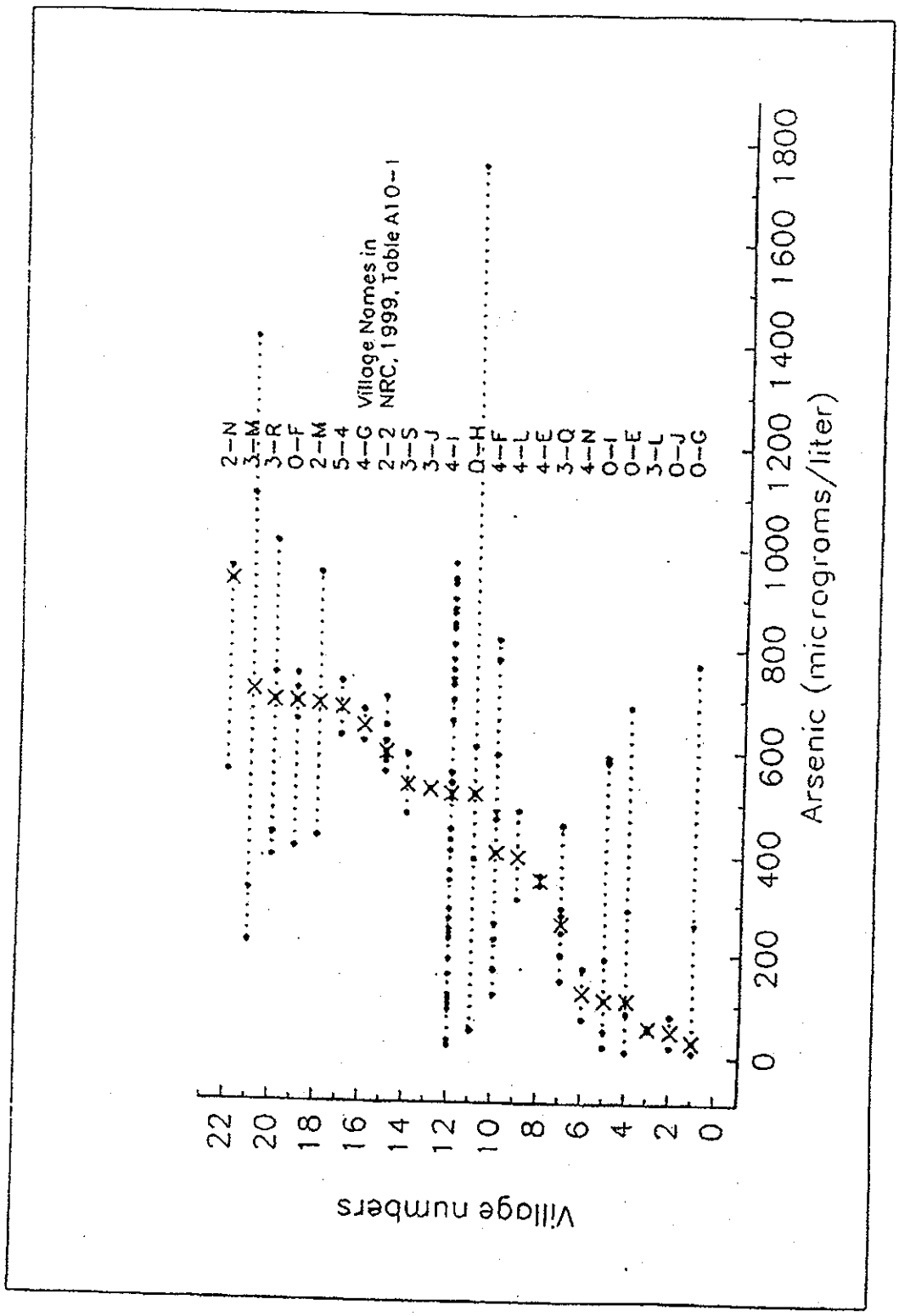

Figure 1. Arsenic well tests for villages with more than one well. (NRC1, Table A10-1)

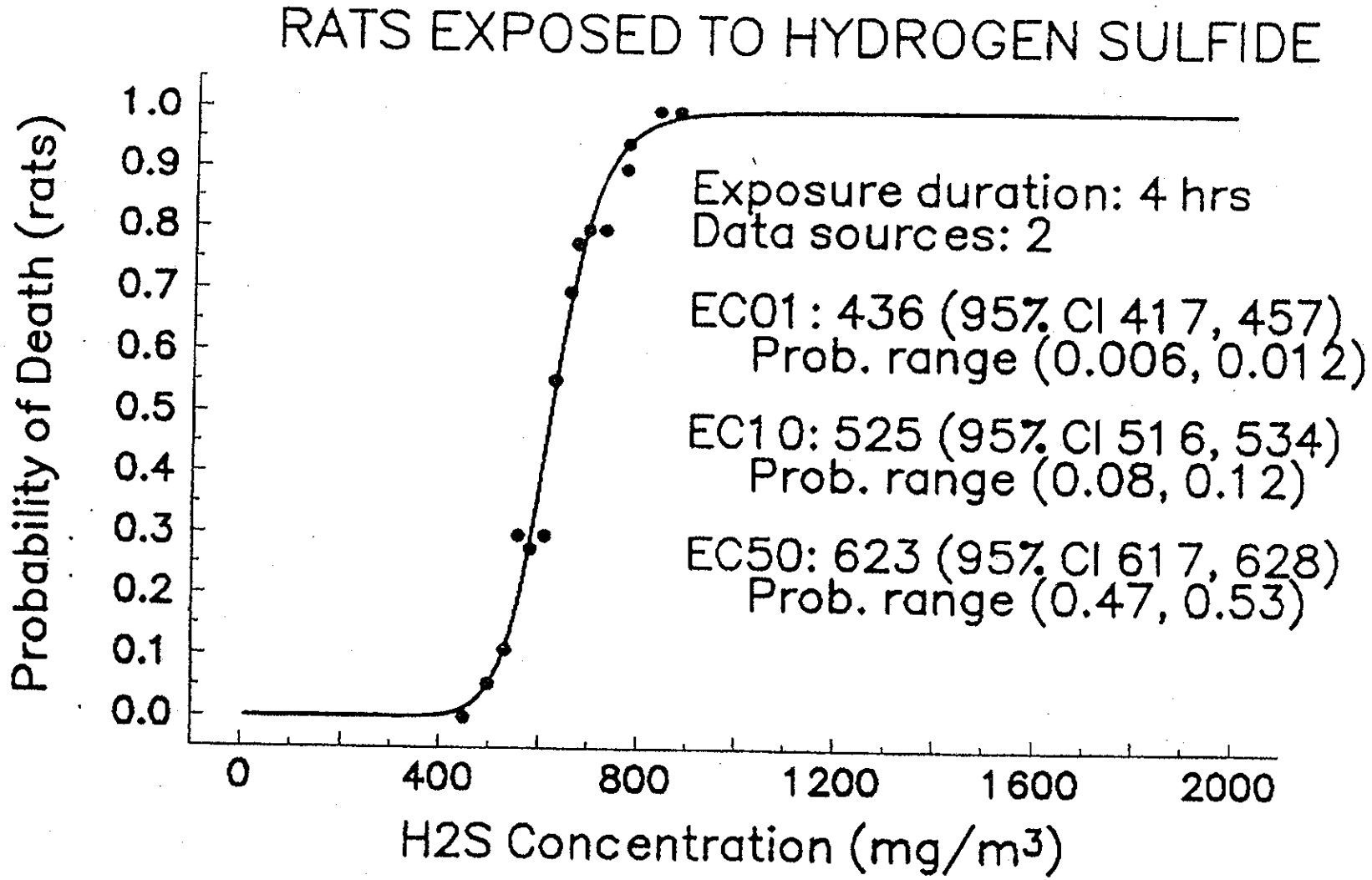

Figure 2. Example of dose-response (mortality of rats exposed to hydrogen sulfide fit with by logistic regression)

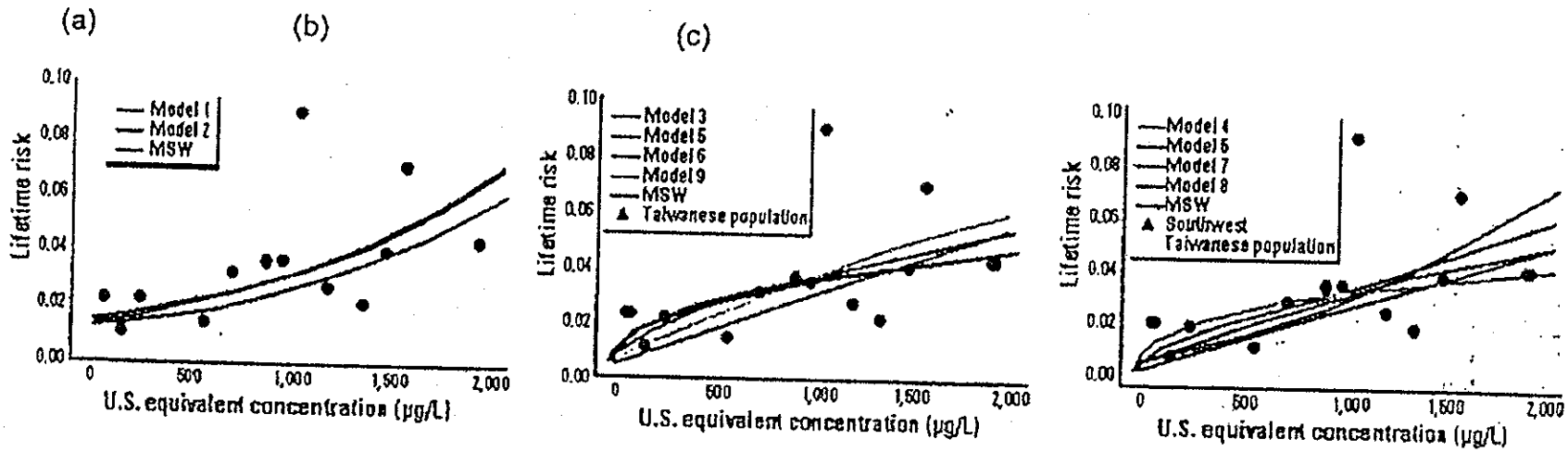

Figure 3. Estimated lifetime death risk over background rates in Taiwan for male bladder cancer (a) without comparison population, (b) with Taiwanese-wide population, (c) with southwestern Taiwanese region comparison population. Morales et al. (2000). (Reprinted with permission of Environ. Health Perspect.)

Memorandum

To: CPSC Commissioners **Date:** February 27, 2003

From: Barbara D. Beck, PhD

Subject: Overview of comments regarding CPSC's risk assessment of CCA-treated playground equipment

This memorandum provides an overview of my comments regarding the Briefing Package pertaining to the Petition to Ban Chromated Copper Arsenate (CCA)-Treated Wood in Playground Equipment (Petition HP 01-3) prepared by the staff of the U.S. Consumer Product Safety Commission (CPSC, 2003). I will present my comments orally to the CPSC Commissioners on March 17, 2003, and in writing by March 28, 2003.

Certain assumptions used by CPSC staff regarding the assessment of exposure to dislodgeable residue and the toxicity of arsenic likely result in an overestimate of exposure and risk. The combined effect of these assumptions may well overestimate the staff's high-end cancer risk by 10- to 20-fold, meaning that the maximum estimated cancer risk could be as low as 5×10^{-6}, and the low-end estimated cancer risk could be about 6×10^{-7}. In addition, by presenting certain alternative assumptions that are implausibly high, the sensitivity analysis provides a false sense of confidence that the resulting estimates are conservative, yet realistic. It should also be noted, that because of a lack of information in the staff report we were unable to evaluate certain aspects of the report, such as the QA/QC procedures, which are critical for establishing confidence in the data.

The following bullets list a few examples of parameters that are likely to overestimate exposure or risk in the staff report. Other examples will be provided in the oral presentation and in the written comments.

- Arsenic cancer slope factor (CSF). The risk assessment uses a range of CSFs from 0.00041 µg/kg-day (0.41 mg/kg-day) to 0.023 µg/kg-d (23 mg/kg-day). The lower end of the range is from the USEPA's 2001 risk assessment for arsenic in drinking water, as part of the Agency's MCL analysis (CFR 66(14), January 22, 2001, USEPA, 2001), whereas the higher end of the range is derived from the National Research Council's study of arsenic in drinking water (NRC, 2001). The upper end of the range is implausible and inconsistent with the results of well designed studies in U.S.

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communities with elevated concentrations of arsenic in drinking water (Lewis *et al.*, 1999; Frost *et al.*, 2002). In addition, this CSF value is inconsistent with the methodology used by the USEPA in its MCL risk assessment.

- Relative bioavailability of dislodgeable arsenic. The risk assessment assumed that the relative bioavailability of arsenic in dislodgeable residue is the same as that of arsenic in drinking water. There is no basis for assuming that this material is highly soluble. In fact, data from leaching studies of CCA-treated wood (CPSC, 1990; Cooper, 1991; Murphy and Dickson, 1990; Osmose, 2000; Warner and Solomon, 1990), solubility studies of dislodgeable residue (Cui, 2001; Osmose, 2001), and a pilot feeding study in hamsters (Aposhian, 2001) indicates that the bioavailability of dislodgeable arsenic is likely to be significantly less than 100%, and could well be below 50%.
- High-end hand transfer efficiency factor (HTE). The risk assessment assumed that it was possible that the HTE could be as high as 7.0, meaning that the total residue on the hands could be loaded and unloaded and completely ingested 7 times per day. This is implausible and not consistent with the underlying studies on which the HTE is based. Bounds can be placed on the HTE through the use of alternative soil ingestion and hand loading rates, as was done in the Gradient risk assessment of CCA-treated wood (Gradient, 2001). In this analysis, the maximum plausible HTE was estimated as 1.0.

In addition, the staff report fails to provide a balanced picture of risk by not expanding on the discussion of the estimated potential intake of arsenic from treated wood in comparison to intakes of arsenic from other sources. For example, the staff report notes that the estimated intake of arsenic would be at the low end of the range of arsenic intake from food (*i.e.*, naturally occurring arsenic). However, the report neglects to mention that the intake from treated wood would have a modest impact on total exposure to arsenic. Moreover, the report should discuss how the estimated intake of arsenic (setting aside the issue of its validity) is less than what is permitted under the USEPA's drinking water standard for arsenic, and less than what has been permitted at several sites remediated under the USEPA's Comprehensive Environmental Response Conservation and Liability Act (USEPA, Region 10, 1993; USEPA, 1998). Such comparisons are critical to accurately communicate the significance of the staff report's exposure and risk estimates to risk managers and to the public.

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Comments by Joyce S. Tsuji, PhD, DABT
Exponent
On Behalf of the Wood Preservative Science Council
2/28/03

Children's exposure to arsenic from treated wood playsets is well within the range of background exposures and below lifetime background exposure to arsenic

Arsenic is a natural component of our environment, and is known to be ubiquitous in soil, water, and in the diet. Consequently, understanding where arsenic exposures fall with respect to natural background is important for communicating the relative magnitude of the risk in perspective with everyday exposures. The relation of exposure to background determines whether the estimated risks are purely hypothetical based on extrapolation of data at high doses to low doses, or are in excess of normal doses and therefore, depending on the magnitude of this excess, potentially may result in an observable increase in disease in a population.

Background sources of arsenic to the general population include food, water, and soil. The following example calculations provide additional evidence that background doses from food and water are considerably higher than CPSC's calculated exposure from CCA-treated playsets.

As recognized by the CPSC, the diet is the primary source of arsenic exposure for the general population. Arsenic is not added to food, but is present naturally. Background arsenic exposure for young children as noted by CPSC ranges from about 2 to 46 ug/day (including perhaps some organic as well as inorganic forms of arsenic). A recent evaluation of dietary inorganic arsenic intake in children ages 2 to 5 reports a mean of 3.2 ug/day with a 99th percentile of 9.4 ug/day (Yost et al. 2002).

Arsenic is present naturally in an inorganic form in groundwater, and in several parts of the United States exceeds the new lower MCL level of 10 ug/L. The calculations of dietary intake of Yost et al. (2002) were based on a water arsenic concentration of 0.8 ug/L. For populations with higher concentrations of arsenic in water, the dose associated with the diet (including drinking water and water used for cooking) would increase accordingly. For example, drinking water doses associated with a 1 to 10 ug/L concentration of arsenic in water are 1 to 10 ug/day, assuming a drinking water intake for a child of 1 L/day. Background food and water exposure to inorganic arsenic at the new MCL level would thus be about 13 ug/day on average with a 99th percentile close to 20 ug/day.

To compare these background doses primarily from water and food to inorganic arsenic exposure to CCA-treated playsets, the dose from playsets (3.3 ug/day according to the CPSC calculations, "about 3.5 ug/day" according to the CPSC fact sheet) should be corrected for the number of days per year a child plays on a playset (estimated by CPSC to be 156 days/year). This results in a calculated average dose of 1.4 ug/day for exposure

to playsets over a year. CPSC's estimate of the contribution of inorganic arsenic from contact with treated wood playsets is thus a fraction of background exposures to inorganic arsenic from diet and water.

For the purposes of comparing to background arsenic cancer risks, these calculated childhood doses should be converted to a cumulative dose averaged over a lifetime. Early childhood exposure (when mouthing behavior is highest) to CCA-treated playsets is limited to 5 years in the CPSC risk assessment, whereas exposure to arsenic in diet and water continue for a lifetime. Exposure to playsets for only 5 out of 70 years would result in a cumulative lifetime average daily dose of 0.1 ug/day. The cumulative lifetime inorganic arsenic dose or risk associated with CCA-treated playsets would thus be considerably less than that for lifetime exposure to inorganic arsenic in water and diet.

These calculations indicate that even though CPSC has calculated hypothetical cancer risk estimates for children playing on CCA-treated wood, the magnitude of exposures and associated risks from playsets are well below the range of exposure and risk from background exposure to inorganic arsenic. Therefore, these exposures would have little effect on any lifetime risk associated with inorganic arsenic. The cancer risk estimated by CPSC is thus a hypothetical small risk rather than one that would actually increase the background risk associated with arsenic in the population.

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Oral Presentation of Floyd Frost, Ph.D., Epidemiologist, Lovelace Respiratory Research Institute (LRRI), 2425 Ridgecrest SE, Albuquerque, NM 87108, 505-348-8776, email: ffrost@lrri.org

Filed on behalf of the Wood Preservative Science Council in response to: March 17, 2003 "Public Briefing on CCA Pressure-Treated Wood Playground Equipment Scheduled" issued February 7, 2003.

I am an epidemiologist with 13 years of applied public health experience at the Washington State Department of Health and 12 years of research experience at LRRI. I have worked on arsenic issues in relation to EPA regulation of arsenic emissions from the ASARCO Ruston copper smelter near Tacoma, Washington (1980-83) and as a consultant for the City of Albuquerque regarding the 2002 revision of the arsenic maximum contaminant level for drinking water. I have served on arsenic-related advisory panels for the EPA and for the State of New Mexico. I have published several papers on arsenic-related health effects.

The arsenic-related lung and bladder cancer risk estimates used by the Consumer Product Safety Commission (CPSC) staff are based on extrapolations from the Taiwan and South American data for high dose exposures. The extrapolation method was developed from policy assumptions that dose and response have a linear relationship (i.e. a proportional increase in risk with increased dose) rather than from knowledge of the biological mechanisms. This is not even considered by the EPA to be the most plausible relationship between arsenic exposure and cancer risks (US EPA, 2000, p.38901), (Snow et al., 2000), (Menzel et al., 2000). Therefore, the risk estimates derived are, at best, highly uncertain. The Taiwan and South American studies used in making the EPA and CPSC risk extrapolations used populations with extremely high arsenic exposures. Yet the best available data from the published epidemiological studies of U.S. and European populations exposed to low or moderate arsenic levels (Lewis et. al., 1999; Bates et al., 1995; Kurttio et al., 1999; Burchet and Lison, 1998) show little or no indication that the lung and bladder cancers risks are increased. Although the arsenic concentrations in these studies are lower than those received by the Taiwan population, they are much higher than would be expected from exposure to pressure treated wood used in playground equipment. Since the focus of the proposed regulation is very low-dose arsenic exposures, greater reliance should be placed on extrapolating from the more relevant low-dose U.S. and European studies.

In addition to the research cited by EPA, we have recently completed a project that is relevant to childhood arsenic exposures. We followed a cohort of children to determine if childhood exposure to high ambient air arsenic is associated with increased cancer mortality risks. The results were presented at the 2002 Society for Epidemiology Research Meeting in Palm Desert, California (Tollestrup et al. 2002). This study followed 1,827 boys and 1,305 girls residing near the ASARCO Ruston copper smelter from 1907 to 1932. Lung and bladder cancer mortality risks through 1985 among members of the highest exposure groups were compared to risks for members of the lowest exposure groups. We used a Cox proportional hazard model. It showed no

evidence of elevated bladder or lung cancer mortality risks in the highest three arsenic exposure categories. Arsenic exposure levels for this cohort are uncertain, but in 1976 after emission controls were added to the smelter to reduce exposure levels, arsenic exposure averaged close to 3 micrograms per cubic meter of air in the town of Ruston. Average urinary arsenic levels for the mid 1970's averaged 60-150 ppb compared to 10-50 ppb for children residing distant from the smelter. ASARCO significantly improved their arsenic recovery in the 1930's and 1940's, suggesting that exposures during the 1907-32 period were much higher than those measured in the 1970s. In all cases, however, it should be noted that arsenic exposure from a smelter presents a far different, and potentially more serious, case than any potential exposure from treated wood.

We are also completing a study of mortality from arsenic-related cancers in U.S. counties with elevated drinking water arsenic levels. This study examines 50 years of lung and bladder cancer mortality and compares mortality rates between counties with high, medium and low drinking water arsenic levels. This study will be completed in six months.

In addition to concerns about the risk estimates used, we are also concerned about the exposure estimates. The studies cited by the CPSC in support of their exposure estimates are not described, are not available to the reader and have not been subject to scientific peer review. Since the estimated risks depend greatly on the dose estimates, it is critical that the methods for estimating exposure be subject to peer review and made available to the public. In addition, no actual human exposure estimates have been conducted to validate any of the exposure estimates made by the CPSC.

We believe that both the risk and exposure estimates should be based on the best available science that is subject to external review. In particular, all aspects of the deliberation should be made available to the public and open to review.

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AMERICAN FOREST & PAPER ASSOCIATION

TESTIMONY BEFORE CONSUMER PRODUCT SAFETY COMMISSION ON CCA BAN PETITION, HP 01-3
March 17, 2003

Good morning. I am Sharon Kneiss, vice president for regulatory affairs of the American Forest & Paper Association (AF&PA). Thank you for this opportunity to testify on the wood preservative CCA, chromated copper arsenate.

AF&PA is the national trade association of the forest products industry. We represent more than 200 companies and related associations that engage in or represent the manufacture of pulp, paper, paperboard and wood products. The forest products industry accounts for about 7 percent of total U.S. manufacturing output, employs 1.5 million people and ranks among the top ten manufacturing employers in 42 states.

AF&PA members produce much of the softwood used to manufacture treated lumber. Treated wood comprises an $8 billion annual retail market.

We believe it is not necessary for the Commissioners to act on the petition to ban CCA-treated wood in playground equipment because that result has already been achieved. As you know, the Environmental Protection Agency (EPA) announced a year ago that "the manufacturers of CCA had requested that registration of CCA be either cancelled or amended to terminate essentially all residential uses of CCA, including use in playground equipment, effective Dec. 31, 2003. Therefore, no useful purpose would be served by further action on CCA by CPSC.

Moreover, we believe that any regulatory action should be based on sound science. If the Commissioners are not inclined to deny the petition now, they should at least defer action on the petition until:

- EPA acts on the registrants' request (as CPSC staff recommends),
- Additional data collection is completed,
- EPA completes its risk assessment, and
- The joint EPA/CPSC exposure mitigation study is completed.

Regrettably, we believe that the staff briefing paper has reached conclusions about risk without complete information, thereby creating unnecessary concern among parents and confusion in the treated wood marketplace.

In addition, CPSC staff's premature risk conclusions differ from those issued by EPA. Administrator Whitman, said, when announcing the voluntary industry transition to the next generation of wood preservation chemicals, "EPA has not concluded that CCA-treated wood poses unreasonable risks to the public for existing CCA-treated wood being used around or near their homes or from wood that remains available in stores."

She has also made clear to the public that there is no need to disturb existing CCA- treated structures or surrounding dirt. Specifically, Administrator Whitman said, "EPA does not believe there is any reason to remove or replaced CCA-treated structures, including decks or playground equipment. EPA is not recommending that existing structures or surrounding soils be removed or replaced."

CPSC staff conclusions also differ from those of the Florida Physicians Arsenic Workgroup, an expert panel of physicians appointed by the Florida Department of Health. This panel concluded after a year of study that CCA-treated wood is safe for use in playgrounds and recreational facilities. The panel's report to the Florida Secretary of Health states:

"The purpose of this review was to evaluate the risk of clinical disease associated with the use of CCA-treated wood for construction of playground equipment and recreational facilities. The available data have not demonstrated any clinical disease associated with arsenic exposure from this use of the CCA-treated wood. In addition, there have been no reported clinical cases of arsenic-induced manifestations that would be concordant with an excessive exposure to arsenic contaminated soil resulting from use of CCA-treated wood at playground and recreational facilities. ....

"Therefore, the Physicians Arsenic Work Group agrees with and supports the United States Environmental Protection Agency's directive that 'EPA does not recommend consumers replace or remove existing structures made with CCA-treated wood or the soil surrounding those structures.'"

Federal and state governments should speak with one voice about the science surrounding CCA and about wood structures treated with that material. Otherwise, the potential for public confusion is substantial and confidence in government bodies charged with protecting the public with science-based decisions will be weakened.

False or premature alarms are as damaging to that confidence as the failure to address real risk. The registrant's voluntary actions already achieve the remedy requested by the petition; there is no need to act on incomplete information.

For these reasons we urge the Commissioners to deny the petition, because no useful, practical purpose will be served by CPSC action after EPA soon approves termination of CCA manufacture to treat lumber used for residential purposes, including playground equipment. Failing that, the Commissioners should insist on good, complete science before taking any action on the staff recommendation.

Thank you and I'll be happy to take your questions.

Florida Physicians Arsenic Workgroup

June 14, 2002

John Agwunobi, M.D.
Secretary of Health, State of Florida
Florida Department of Health
Tallahassee, Fl

Dear Dr. Agwunobi:

We have conducted an extensive review of the medical literature concerning the toxicity and carcinogenicity of arsenic, its environmental and natural occurrence, bioaccessibility and bioavailability, and past medical uses. The purpose of this review was to evaluate the risk of clinical disease associated with the use of CCA treated wood for construction of playground equipment and recreational facilities. The available data have not demonstrated any clinical disease associated with arsenic exposure from this use of the CCA treated wood. In addition, there have been no reported clinical cases of arsenic-induced manifestations that would be concordant with an excessive exposure to arsenic contaminated soil resulting from use of CCA treated wood at playground and recreational facilities. The physical-chemical properties of arsenic and the methods of production of CCA treated wood prevent a significant exposure from the ordinary and customary use of playground equipment and recreational facilities constructed with this material.

Used since the 1960s, CCA-treated wood has never been linked to skin diseases or cancer in children exposed during recreational use. Manifestations of arsenical skin diseases and cancers would be expected after 30+ years of use if toxic levels of arsenic were leaching from the wood. Thus, the levels of arsenic in or around CCA-treated wood in playgrounds and recreational facilities does not appear to be sufficient to adversely affect the health of children or adults.

Based on a review of the mechanisms of bioaccessibility and bioavailability of arsenic from soil and CCA treated wood as would occur around playgrounds and recreational facilities, the bioavailability of arsenic from CCA treated wood is low. Furthermore, the concentrations of arsenic found in Florida soils are as much as two orders of magnitude lower than acceptable background levels for other parts of the United States. Thus, increases in soil arsenic levels may appear to be elevated at some playground and recreational facilities, but are similar to background levels in other parts of the country. Therefore, the Physicians Arsenic Work Group agrees with and supports the United States Environmental Protection Agency's directive that "EPA does not recommend

consumers replace or remove existing structures made with CCA-treated wood or the soil surrounding those structures " .

The potential risks associated with exposure to arsenic-containing soil or wood products are determined by the bioaccessibility and bioavailability of arsenic. Since the bioavailability of arsenic from playground and recreational wood and soil is low, the amount that could be absorbed also remains low and helps to further explain the absence of arsenic toxicity cases associated with the use of playground and recreational structures containing CCA treated wood.

The Physicians Arsenic Work Group does not recommend sampling of playground and recreational areas containing customarily used CCA treated wood. This conclusion is based upon previous deliberations concerning the toxicity and bioavailability of arsenic. Further, low levels of arsenic, less than 50ppm, are found naturally in many areas of the United States. Finally, the amount of arsenic that could be absorbed from playground soil and CCA treated wood is not significant compared to natural sources and will not result in detectable arsenic intake.

We thank you for your leadership in addressing this important issue.

Sincerely yours,

Pascual Bidot, M.D., MSPH

M. Rony Francois, M.D., MSPH

Joette Giovinco, M.D., MPH

Jim Hillman, M.D.

David Johnson, M.D., MPH

Howard Weiner, M.D., MPH

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Whitman Announces Transition from Consumer Use of Treated Wood Containing Arsenic

February 12, 2002

EPA Administrator Christie Whitman today announced a voluntary decision by industry to move consumer use of treated lumber products away from a variety of pressure-treated wood that contains arsenic by Dec. 31, 2003, in favor of new alternative wood preservatives. This transition affects virtually all residential uses of wood treated with chromated copper arsenate, also known as CCA, including wood used in play-structures, decks, picnic tables, landscaping timbers, residential fencing, patios and walkways/boardwalks. By Jan. 2004, EPA will not allow CCA products for any of these residential uses.

"This action will result in a reduction of virtually all residential uses of CCA-treated wood within less than two years," said EPA Administrator Christie Whitman. "Today's announcement greatly accelerates the transition to new alternatives, responding to market place demands for wood products that do not contain CCA. This transition will substantially reduce the time it could have taken to go through the traditional regulatory process."

"This is a responsible action by the industry," Whitman continued.

"Today's action will ensure that future exposures to arsenic are minimized in residential settings. The companies deserve credit for coming forward in a voluntary way to undergo a conversion and retooling of their plants as quickly as possible. The transition to new alternatives will provide consumers with greater choice for their building needs."

The transition period will provide consumers with increasingly more non-CCA treated wood alternatives as the industry undergoes conversion and retooling of their industrial equipment and practices, while also allowing adequate time to convert treatment plants with minimal economic disruption for the industry's employees. Beginning immediately, and over the next 22 months, wood treatment plants will convert to new alternative wood preservatives that do not contain arsenic. In the current year, manufacturers expect a decline in production of CCA products for affected residential uses up to 25 percent, with a corresponding shift to alternatives. During 2003, the companies expect the transition away from CCA to continue and increase, with a decline in production of CCA products for affected residential uses up to 70 percent, with a corresponding shift to alternatives. New labeling will be required on all CCA products, specifying that no use of CCA will be allowed by the wood-treating industry for the affected residential uses after Dec. 31, 2003.

EPA has not concluded that CCA-treated wood poses unreasonable risks to the public for existing CCA-treated wood being used around or near their homes or from wood that remains available in stores. EPA does not believe there is any reason to remove or replace CCA-treated structures, including decks or playground equipment. EPA is not recommending that existing structures or surrounding soils be removed or replaced.

While available data are very limited, some studies suggest that applying certain penetrating coatings (e.g., oil-based semi-transparent stains) on a regular basis (one re-application per year or every other year depending upon wear and weathering) may reduce the migration of wood preservative chemicals from CCA-treated wood.

Arsenic is a known human carcinogen and, thus, the Agency believes that any reduction in the levels of potential exposure to arsenic is desirable. As always, when children play outside, whether around CCA-treated play structures or not, they should wash their hands prior to eating. Also, food should not be placed directly on any outside surface, including treated wood. CCA-treated wood should never be burned, as toxic chemicals may be released as part of the smoke and ashes. Consumers who work with CCA-treated wood are encouraged to use common sense in order to reduce any potential exposure to chemicals in the wood. Specific actions include sawing, sanding and machining CCA-treated wood outdoors, and wearing a dust mask, goggles and gloves when performing this type of activity. Clean up all sawdust, scraps and other construction debris thoroughly and dispose of it in the trash (i.e., municipal solid waste). Do not compost or mulch sawdust or remnants from CCA-treated wood. Those working with the wood should wash all exposed areas of their bodies thoroughly with soap and water before eating, drinking or using tobacco products. Work clothes should be washed separately from other household clothing before wearing them again.

Chromated copper arsenate, or CCA, is a chemical compound mixture

containing inorganic arsenic, copper and chromium that has been used for wood preservative uses since the 1940s. CCA is injected into wood by a process that uses high pressure to saturate wood products with the chemicals. CCA is intended to protect wood from dry rot, fungi, molds, termites, and other pests that can threaten the integrity of wood products.

During the past several months, CCA-treated wood has been the subject of an EPA evaluation under provisions of the Federal Insecticide, Fungicide, and Rodenticide Act, which direct EPA to periodically reevaluate older pesticides to ensure that they meet current safety standards. The Agency is continuing to proceed with a risk assessment. EPA is also continuing to evaluate public comments and input from an external scientific review panel on methodologies to perform a risk assessment for residential settings and potential exposure to children from CCA.

More information on this announcement is available at www.epa.gov/pesticides/citizens/1file.htm.

This page originally created: Tuesday, February 12, 2002

SOUTHERN PINE COUNCIL

TESTIMONY BEFORE CONSUMER PRODUCT SAFETY COMMISSION
ON CCA BAN PETITION, HP 01-3
March 17, 2003

Good morning. I am Debbie Burns, vice president for public affairs of the Southeastern Lumber Manufacturers Association (SLMA). Today I am testifying on behalf of the Southern Pine Council – a joint program supported by SLMA and the Southern Forest Products Association. Thank you for this opportunity to testify on the wood preservative CCA, chromated copper arsenate.

The Southern Pine Council represents Southern Pine manufacturers. In 2002, Southern Pine production was 16.5 billion board feet and 7.29 billion board feet of that total -- 44 percent -- was treated. The vast majority - nearly 85 percent - of all treated wood is Southern Pine. As the largest single use category for Southern Pine lumber, treated wood is critical to our manufacturers. Treated Southern Pine comprises an $8 billion annual retail market.

We believe it is not necessary for the Commissioners to act on the petition to ban CCA-treated wood in playground equipment because that result has already been achieved. As you know, the Environmental Protection Agency (EPA) announced a year ago that "the manufacturers of CCA had requested that registration of CCA be either cancelled or amended to terminate essentially all residential uses of CCA, including use in playground equipment, effective Dec. 31, 2003."

In addition, the playground equipment manufacturers have already reacted to market demand by moving to products treated with alternate preservatives. Most major playground equipment manufacturers have already transitioned away from CCA-treated wood. Our members who sell to playground equipment manufacturers started reporting a shift away from the use of CCA-treated wood even prior to the February 2002 announcement from EPA. Therefore, no useful purpose would be served by further action on CCA by CPSC.

We believe any action on the petition to ban would unnecessarily disrupt the marketplace and place the treated wood industry in further jeopardy. The majority of wood treaters are small business manufacturers who are already burdened by the capital investment needed to change to new generation preservatives.

A premature report by the CPSC could raise consumer alarm over an issue that has already been dealt with in the marketplace in regards to playground equipment and will be dealt with on all other residential applications by the end of the year.

In summary, the Southern Pine Council urges the Commissioners to deny the petition, because no useful, practical purpose will be served by CPSC action. Absent a denial of the petition, the Commissioners should insist on sound, complete science and a coordinated effort with the EPA before taking any action on the staff recommendation.

Thank you and I'll be happy to take your questions.

Request To Make Oral Presentation
March 17, 2003 Public Meeting
Consumer Product Safety Commission

Regarding: Petition HP 01-3, Chromated Copper Arsenate Treated Wood In Playground Equipment

Affiliation: I am Vice President of Wood Preservation for Universal Forest Products, Inc. Subsidiaries of Universal Forest Products, Inc., collectively "UFP," produce pressure treated wood products.

Presentation Text

UFP is a treater buying preservatives (including CCA and ACQ) from the pesticide registrants and lumber from mills, manufacturing pressure treated wood products and selling them to primarily retailers. UFP considers actions already taken by our industry equivalent to "voluntary standards" encouraged by CPSC. These include an enhanced Consumer Awareness Program and transitioning to alternative preservatives for essentially all consumer uses including playground equipment. Further, it has been UFP's experience that playground equipment manufacturers whom we sold treated wood switched to alternative treated wood products more than a year ago. UFP is concerned with the confusion and uncertainty created for the general public by the CPSC action. UFP considers Petition HP 01-3 moot. Actions already taken by the industry address the concerns raised in the petition and further CPSC action does nothing to actually protect the public.

Scott W. Conklin
February 28, 2003

Text of Oral Presentation

Hal M. Storey
Vice-president
S.I. Storey Lumber Co., Inc.
A lumber manufacturing plant that includes pressure treating processes

March 17, 2003

Public hearing on The CPSC Staff Briefing Paper
RE: Petition HP 01-3

I appreciate the opportunity to be here and to join others offering comments on petition HP 01-3, a petition for your commission to enact a ban on use of CCA treated lumber in playground equipment.

I am one of a third generation involved in running our company. We are a family owned small business that has been a manufacturer of untreated pine lumber for 83 years. In addition, we have been a manufacturer of CCA treated pine products for more than 32 years without incident.

During the time we have produced CCA treated lumber, we are fortunate in that our customer base has included manufacturers of commercial playground structures. Our current customer base still includes several of these manufacturers.

During the time we have had these playground manufacturing accounts, I can tell you that we know of no known claims as a result of CCA treated lumber in playgrounds. In addition, all of our current playground-manufacturing customers have converted to alternative treated products – products other than CCA.

Of these manufacturers in our customer base, some of them made the conversion to treated products other than CCA well in advance of the label modifications announced by the registrants and the EPA. Others made the conversion early in 2002. Regardless of when our customers made their business decision to convert to other preservatives, they did so because the market demanded it, not because of any problems with the existing products.

Therefore, based on our long-history with CCA treated lumber and our current knowledge of the playground marketplace, it is our contention that the petition is unnecessary due to the voluntary label modifications announced by the registrants of CCA and EPA as a result of the re-registration process. Furthermore, it is our belief that the warnings associated with the release of the staff's report were issued prematurely due to incomplete science and incomplete EPA assessments. In addition, it is our belief that the warnings and the ban petitioned for are unnecessarily alarming to the public and harmful to industry without scientifically proven basis.

In conclusion, I respectfully ask and support the commission's denial of this petition because it is unnecessary. In the absence of an outright denial by you, I respectfully ask you to delay any further action until action is proven to be needed.

Thank you.

February 28, 2003

Summary of Presentation of Seth Goldberg, Steptoe & Johnson LLP
On Behalf of the Wood Preservative Science Council

The Wood Preservative Science Council (WPSC) represents registrants of chromated copper arsenate (CCA) wood preservative. The WPSC urges the Commission to deny the Petition. There are a wide range of scientific and common sense reasons to take this action, the most important of which is that the Petition is moot.

The Petition seeks a ban on the use of CCA treated wood to manufacture children's play structures. That already has been accomplished by the voluntary cancellation of certain uses that registrants have sought from EPA. The voluntary cancellation is set to become effective at the end of this calendar year, and the transition to alternative products for children's play structures is already well under way in the marketplace. Since the only relief sought by the Petition has been accomplished by other means, the Commission has no logical choice but to deny the Petition.

In addition, it is important for the Commission to recognize EPA's current effort to determine whether any regulation is necessary in this area. EPA has been engaged on the issue of CCA used in children's play structures since early 2001. It has convened two Science Advisory Panels to obtain the views of experts on exposure and risk assessment issues. To fill data needs identified by these independent science advisors to EPA, WPSC members are conducting over $1.5 million in additional scientific research, which EPA has announced it will use in conducting a risk assessment later this year.

Because EPA is currently engaged in addressing this issue, no purpose is served by duplicative action by the Commission. Any action but denial of the Petition can only serve to

confuse the public and cause the CCA Registrants to expend resources that could be better deployed elsewhere.

Finally, it is important to point out several serious concerns with the process the Commission is following in this matter.

First, as explained in comments filed by the American Chemistry Council Biocides Panel Arsenical Wood Preservative Task Force in response to the original Petition, the Commission does not have jurisdiction over CCA treated wood because EPA has exercised jurisdiction. In fact, EPA has asked for additional science to be generated by WPSC members, and plans to perform a risk assessment of the use of CCA treated wood for playground equipment once the additional data are in. Therefore, in denying the Petition, the Commission should acknowledge EPA's jurisdiction.

Second, WPSC and other interested parties have not had an adequate opportunity to analyze the Staff Report before submitting these written statements, and, absent a delay, will not have such an opportunity before the public meeting. The Commission also has failed to provide any of the scientific back-up information or peer reviews. This makes it impossible for WPSC and other parties to fully evaluate the Staff Report and the conclusions it makes based upon that scientific information. WPSC strongly believes that good government requires sound science and public review. The Commission should promptly make all requested background materials available and provide an adequate opportunity for interested parties to evaluate and address this information before proceeding.

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Comments of Angela Logomasini
Director of Risk and Environmental Policy, Competitive Enterprise Institute
Before the Consumer Product Safety Commission
Regarding the use of Chromated Copper Arsenate in Playground Equipment
March 17, 2003

On the surface, the Consumer Product Safety Commission's (CPSC) role in the debate over wood treated with the preservative chromated copper arsenate seems limited. It is currently focused on the question of whether to initiate a regulatory action to answer a petition requesting a ban on the use of CCA on playground equipment. Yet, CPSC has a responsibility beyond this decision before us today. All CPSC activities and studies will impact other regulatory actions, particularly those ongoing at the Environmental Protection Agency (EPA). Accordingly, as a member of the public, I urge that the Commission consider all the implications of its actions and ensure that it does not do needless harm to public health and well being by failing to see the bigger picture.

In particular, the EPA continues to consider what residential uses of CCA-treated wood it will allow after it completes its risk assessment on the issue. In February 2002, EPA stated its intent to ban all residential uses (many don't even have much relevance to children), which included several uses that most people would not consider "residential," such as some agricultural uses. While existing registrants may want to stop selling this product, should EPA reaffirm its long-held position that CCA does not pose an unreasonable risk, it could leave open the possibility for other firms to register this product. Or it could broaden the uses allowed even with a limited residential ban, reducing the number of small businesses, taxpayers, and consumers adversely affected. In addition, EPA is considering whether to classify CCA as a hazardous waste.

Numerous individuals and businesses stand to loose from these actions. CPSC needs to consider the full implications of its actions. That includes consideration of the "risk-risk" implications of every action it takes. Everything in life carries risks and if we demand perfect safety, we can end up trading off small risks for big ones. For example, certainly there are accidents on escalators. Yet we don't ban escalators because we know that there would be more accidents on regular stairways. CPSC must be cognizant that there will likely be adverse health and safety implications of its actions on CCA.

- Consider that a CPSC finding that CCA isn't safe (as your study recently suggested), may encourage local governments, daycare centers, and others to tear out playground equipment, as we have seen happen in Florida. Perhaps wealthy communities will be able to rebuild these structures, but what of the poorer communities? Raising the costs of safe playgrounds may well mean that we will have fewer of them (particularly if localities are prompted to remove playgrounds). Will kids in poor, inner-city neighborhoods be safer without safe play areas? CPSC must consider that the absence of affordable safe playgrounds will create real risks that certainly outweigh theoretical risks of CCA-treated wood.

- If CPSC actions build pressure for EPA to ban residential uses of CCA, consumers will pay at least an additional 20-30 percent for decks, retaining walls, and similar outdoor structures. One farmer noted to EPA in comments that when he investigated the cost of fence posts made with the popular alternative product, he learned that it would cost twice as much as posts treated with CCA and these new posts are only expected to last half as long. Hence, for him, that's a quadrupling of costs.

- The alternatives include upgrading decking material to much more expensive, harder woods such as cedar, redwood, or plastic lumber. It might well be worth investigating whether cutting down more redwood forests would be a good environmental policy. We do know that these options are cost-prohibitive for many families and communities, as they can double the cost of decks, playground equipment, and numerous other outdoor structures. Accordingly, regulators assume that the public can switch to one of several other alternative preservatives, yet each product presents its own risks that regulators have not considered.

- For example, the CPSC notes that it has not tested the alternatives and doesn't know whether they are more or less dangerous than CCA. CPSC simply operates on the assumption that they are less dangerous.

- Most of the alternatives use high levels of copper, which corrodes screws, nails and other fasteners. Consumers must use more expensive stainless steels screws, nails, and other fasteners with these products. This aspect of the new products should raise safety concerns as we can expect that some consumers will use the wrong screws and nails, leading to an increase in deck failures and related injuries and deaths.

- The alternative products leach considerable amounts of copper, which if it reaches waterways can prove toxic to fish. I understand that such potentially adverse impacts on wildlife influenced the State of Florida in its decision against switching to the alternative product.

- I have also learned that when Home Depot and Lowes switched to the alternatives, builders wouldn't buy them because of the problems noted above. Home Depot and Lowes are now selling CCA-treated wood until the ban leaves builders no other choice but the buy the other products.

- Small business implications are also considerable. About 350 wood processors would have to retool their shops at a very high cost before switching to alternative preservatives. Because the alternatives require that all machinery be made of stainless steel, retooling is

expensive. Retooling costs range up to a couple hundred thousand dollars for many small firms.

- Local governments will also feel the cost because your study and any subsequent bans will create pressure for them to remove playground sets made with CCA and go to the expense of replacing them. Of course, we will all feel this cost in the form of higher taxes.

- In addition, several companies are facing litigation based on claims about CCA safety. If CPSC issues faulty research, these firms will find it harder to defend against bogus safety claims. We all know who pays the costs of this type of litigation — the consumer. There isn't any solid data convicting CCA, which may be why many litigants are loosing these suits. The problem is, the lawsuits are still expensive to defend and will be more expensive if government studies on the topic are not conducted properly.

- We must also remember that poorly conducted studies will build pressure for bans on other uses of CCA (such as additional agricultural and construction uses). Of course, there may be big costs associated with such bans.

- CPSC actions may also build pressure for EPA to list CCA-treated wood as a hazardous waste, raising costs for everyone from consumers to cities to small businesses. CPSC should not underestimate this possibility and the associated welfare losses as prices for disposal rise. For example, families may keep decks longer — even when the decks begin to deteriorate and become safety hazards — if both the costs of disposing the wood and building a new deck grow too high.

Science & the CPSC Study. The public may suffer reduced safety and heavy costs simply because CPSC produced a study of questionable scientific value. Even if the Commission defers the issue to EPA completely or denies the petition to consider a rulemaking itself, it needs to reevaluate its study.

My organization has arranged for Dr. Kenneth Brown to discuss serious questions about the assumptions of the CPSC study on this issue. He focuses on two key problems with the study. I am sure there are other scientists that will provide comments and note additional problems. CPSC should consider this information and consider revising its study.

The CPSC study says that, given the recent findings of the National Research Council (NRC) reports on arsenic (1999 and 2001), CCA is not safe for use on playground equipment. While the press has made it appear that children are at risk, CPCS should emphasize that it isn't suggesting that children will get sick. CPSC needs to emphasize they are only claiming that there might be a slight increase of cancer risks among older Americans (exposed to arsenic early in life).

CPSC also needs to put the risk that it found in perspective. Science writer Steve Milloy did a good job translating the CPSC-estimated increased cancer risk. As Milloy explained it, if CPSC was correct in all its very conservative assumptions (which is questionable), CCA might increase a person's lifetime risk for lung cancer from "1.01 percent to between 0.012 to 1.120

percent" and for bladder cancer, the increased risk might rise from "about 2 percent to between 2.0002 to 2.01 percent." Placed in this perspective, I don't think many mothers would panic about pressure treated wood, particularly if they were advised that risks would drop dramatically by preventing hand and mouth activity and hand washing.

But as Kenneth Brown's comments indicate, it's likely that CPSC is vastly overestimating exposures. In particular, CPSC changed its long-held position that CCA posed no unreasonable risk because it used data from the NRC reports on arsenic. These reports relied on data from malnourished Taiwanese populations exposed to relatively high levels of arsenic for decades in their drinking water. The relevance of these studies to short-term exposures to trace levels of arsenic early in life here in the United States is highly questionable. In addition, the NRC noted that the data was highly flawed and it too might well be greatly overestimating risks. Add the fact that research is increasingly indicating that arsenic may even be an essential nutrient or at least offer health benefits at low levels.

Also curious is the fact that the CPSC decided to choose a potency factor for arsenic that it notes is 6 to 56 times more potent that what EPA used to set its drinking water standard. Why CPSC decided such a high potency factor also deserves further evaluation or at least a much more convincing explanation. CPSC seems to be operating with the most conservative assumptions about risk even when the NRC noted that the data potentially had already greatly overestimated risks because of overly conservative assumptions.

CPSC needs science that is more grounded in reality, and it needs to recognize that its actions too may create new risks — risks that may far exceed any theoretical risks emanating from trace-levels of arsenic. CPSC should also remember that a poorer society is not a safer or healthier one. If we raise the cost of living, innovation, and entrepreneurship, we won't only have a poorer world; we will have a less safe one as well.

Comments to the Consumer Products Safety Commission
On Pressure-Treated Wood

Submitted February 28, 2003 by Jody Clarke
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My name is Jody Clarke. I am vice president of communications at the Competitive Enterprise Institute here in Washington, D.C., but today I am here speaking to you as a mother.

I'm not only concerned that the federal government is considering a ban on a product that has been safely used for more than six decades; I think it's absolutely outrageous. When people hear the word "arsenic," it conjures up all kinds of images, and I think some groups are trying to use that to their advantage, even though exposure to it in pressure-treated wood, or water for that matter, is extremely small. You, and the Environmental Working Group, are scaring people—and it's completely unnecessary.

As a mother, I am not worried about my son being exposed to the pressure-treated wood that our deck at home is made of, or the playground equipment in our neighborhood. If anything, I worry about my son falling and hurting himself, not about any "phantom" risks from a wood preservative.

Science writer Steve Milloy recently wrote a column about this issue, and he pointed out that quote "there is not the slightest evidence that any child has ever developed cancer from CCA-treated wood." He also pointed out that studies don't show increased health risks for the workers at wood treatment plants and carpenters. If there's a problem, shouldn't we be seeing an increase of cancer in those groups of people?

The increase in the risk of cancer that's been calculated by the Consumer Products Safety Commission—your group—is incredibly small. So small that I find it unbelievable that any action would be taken to ban the product.

I'm sure you're probably aware that studies have been done that show for young smokers who kick the habit by early adulthood, their risk of cancer returns to normal within a few years. It seems to me that would be the same case with children's exposure to CCA-treated wood, and the exposure to that is far less than with smoking.

Groups that support a ban on pressure-treated wood say children are the victims. The real victims are going to be the families, or anyone, who will end up paying 20 to 30 percent more for decks made out of an alternative—and inferior—product, and the wood processors who will be affected by any ban. You could run some people out of business and I think it's a shame.

Working at a public policy group, I probably do know more than the average person about this issue. But I am not a public policy analyst or a scientist; I am here simply as a citizen who is very concerned about what you might do regarding this issue. I'd like to end with that old adage that is so true: If it's not broken, why fix it?

Thank you for listening.